type II cells, direct activation of the effector caspases by caspase-8 is blocked at the level of the effector caspases by IAPs, such as XIAP. For example, the cleavage of BID by caspase-8 is required to release Smac to neutralize the IAPs and allow direct activation of the effector caspases by caspase-8 (*see* Figure 14). Accordingly, by expressing a cytosolic form of Smac, the type II cells should be made sensitive to death receptor-induced apoptosis.

In the Claims:

Please cancel claims 1-27 and 52-96.

REMARKS

Claims 28-51 are pending in the instant application. Claims 1-27 and 52-96 have been cancelled without prejudice to the filing of any continuation, continuation-in-part or divisional applications. The paragraph at line 25, page 45 has been amended to correct a typographical error.

Attached hereto is a marked-up version of the changes made to the specification and claims by the current amendment. The attached page is captioned "**Version With Markings to Show Changes Made.**"

All of the claims remaining in the application are now clearly allowable. Favorable consideration and a Notice of Allowance are earnestly solicited.

Respectfully submitted,

Seed Intellectual Property Law Group PLLC

William T. Christiansen, Ph.D.
Registration No. 44,614

WTC:rap
Enclosure:
Postcard

701 Fifth Avenue, Suite 6300
Seattle, Washington 98104-7092
Phone: (206) 622-4900 Fax: (206) 682-6031

VERSION WITH MARKINGS TO SHOW CHANGES MADE

In the Specification:

Please replace the paragraph beginning at page 45, line 25, with the following rewritten paragraph:

This example discloses that the expression of a cytosolic Smac converts a type II cell cancer to a type I cancer cell. In type II cells, such as breast adenocarcinoma MCF-7 cells, death receptor-induced apoptosis can be blocked by expression of Bcl-2 or Bcl-xL. Whereas, type I cells, such as B lymphoblastoid cell line SKW6.4, are sensitive to death receptor-induced apoptosis even when ~~Bcl-1~~ Bcl-2 or Bcl-xL are expressed. One explanation for this difference is that in type II cells, direct activation of the effector caspases by caspase-8 is blocked at the level of the effector caspases by IAPs, such as XIAP. For example, the cleavage of BID by caspase-8 is required to release Smac to neutralize the IAPs and allow direct activation of the effector caspases by caspase-8 (*see* Figure 14). Accordingly, by expressing a cytosolic form of Smac, the type II cells should be made sensitive to death receptor-induced apoptosis.

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